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DISEASES OF INFANTS AND CHILDREN

JOHN PITCH LANDON, M.D. Editor

LEADING ARTICLES IN THIS NUMBER

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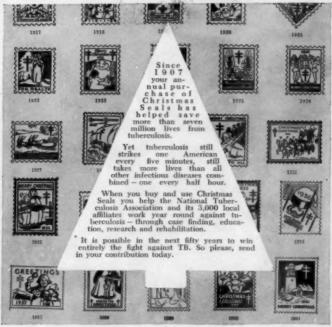
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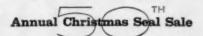
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CONGENITAL STENOSIS OF THE DUODENUM

REPORT OF TWO CASES

FRANK H. RUSS, M.D.*

and

RICHARD E. DUKES, M.D.**
Urbana, Ill.

Congenital stenosis of the duodenum constitute part of the group of congenital anomalies of the gastrointestinal tract characterized by an intrinsic obstruction of that part of the intestinal tract. The literature contains many reports of "intrinsic obstruction" in the newborn in which careful differentiation between the atresias and the stenosis has not been made. This is important, however, as there is considerable difference in prognosis. An atresia denotes a complete obstruction, while a stenosis denotes an incomplete obstruction. The degree of obstruction occurring in congenital duodenal stenosis is reflected in the clinical picture. The majority of duodenal stenosis reported in the literature deal with the more severe forms of obstruction which manifest symptoms very early, usually during the first few hours or days of life. These require immediate surgery, whereas the less severe obstructing lesions may not be evident clinically until later infancy or early childhood and surgery can be delayed.

The two cases reported here are examples of the different degrees of stenosis that may occur. In one, the obstruction did not prevent the infant from growing and developing normally, while in the other the obstruction was such that immediate corrective measures were necessary for the infant's survival.

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**From the Department of Pediatrics, Carle Memorial Hospital and Carle Hospital Clinic, Urbana, Illinois.

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CASE REPORTS

Case 1: L. S., a full-term infant born on February 14, 1952; weight 7 pounds, 2 ounces. Physical examination revealed a normal newborn infant. On February 16, 1952, she started having projectile vomiting after every feeding. Treatment with thick cereal and elixir of phenobarbital, ¼ dram ten minutes before feedings, stopped the projectile vomiting, however, she continued to spit up a little of each feeding. She was discharged from the Hospital on February 23, 1952. During the next three weeks she gained weight steadily but continued to vomit a little of most of her feedings and all of some. She was given a variety of antispasmodics without apparent benefit. X-ray examination of the stomach and small bowel following a barium meal on March 3 showed a partial obstruction at the junction of the second and third portions of the duodenum.

She was checked regularly and did well nutritionally during the next four months in spite of regurgitating large portions of her feedings. Her weight on July 20, 1956 at the age of five months was 16 pounds, 8 ounces. Surgery to relieve the obstruction was performed on July 21, 1952, a retrocolic antiperistaltic duodeno-jejunostomy being done.

Since then she had no vomiting or regurgitation and her growth and development have been normal.

On June 5, 1956, she weighed 42 pounds and physical examination revealed a healthy normal four-year-old girl.

Case 2: K. L. T., a twelve-day-old infant was admitted to Carle Memorial Hospital on August 19, 1953. She had been delivered by cesarean section on August 7, and at birth her weight was 5 pounds, 13 ounces. She had a large amount of mucus and spit up some during the week following delivery; during this period, she had soft green stools at regular intervals. On August 15 she started bringing up bile-stained vomitus, and from that time on, she was unable to retain her formula and the vomiting became projectile.

Physical examination revealed a small infant who did not appear critically ill. The weight was 5 pounds, 1½ ounces; length, 18 inches. The eyes, ears, nose and throat were normal, as were the heart and lungs. The abdomen was negative. Rectal examination was normal.

She was given intravenous fluids and started on elixir metropine with scopolamine, 15 drops ten minutes before feeding. In spite of this therapy, she continued to vomit her formula.

Laboratory examinations on August 19 revealed the following: negative urinalysis; hemoglobin, 16.1 grams per cent; erythrocytes, 4,160,000; and leukocytes, 11,200 with 14 per cent neutrophiles, 80 per cent lymphocytes, 2 per cent monocytes, 2 per cent eosinophiles, and 2 per cent basophiles. The platelets were adequate. X-ray examination of the abdomen showed gas to be distributed

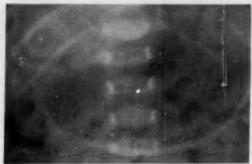


Fig. 1.

throughout the stomach, small bowel and colon (Fig. 1).

On August 21, the infant was given lipiodol by mouth, and it

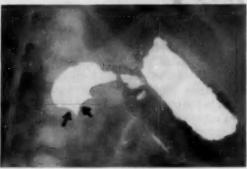


Fig. 2.

was seen to enter the duodenum readily but did not pass into the third portion of the duodenum. Film studies taken in 30 minutes

revealed no lipiodol beyond this point (Fig. 2). After two and one-half hours, only a small amount of lipiodol had passed this point.

A preoperative diagnosis of duodenal stenosis was made and surgery was performed on August 21. Preceding the surgery, a soft rubber catheter was passed through the nose into the stomach. When the abdomen was opened, the catheter was passed distally until the obstruction was encountered within the lumen of the duodenum two centimeters proximal to the ligament of Treitz. The catheter could not be fed past this obstruction. A duodenojejunostomy was done in isoperistaltic fashion between the third part of the duodenum and the jejunum ten centimeters distal to the ligament of Treitz,



Fig. 3.

For the next six days the infant was maintained on intravenous fluids consisting of 10 per cent glucose in water, normal saline, whole blood, and plasma. Even though she was given nothing orally and her stomach was lavaged frequently, she continued to vomit until August 28. At that time her vomiting suddenly stopped and glucose water was started orally. This she retained, as well as a dilute evaporated milk formula. Her feedings were gradually increased and she was discharged from the hospital on September 7. She weighed 5 pounds, 12 ounces, and was taking 3 ounces of an evaporated milk formula every three hours.

She was seen on August 9, 1954 at the age of twelve months. She had done well and had no vomiting.

Physical examination at this time revealed a well developed, well nourished infant weighing 20 pounds. The eyes, ears, nose

and throat were normal. The chest was clear, the heart was normal, and the abdominal examination was negative. X-ray examination of the stomach and small bowel after barium showed the first and second portions of the duodenum to be moderately dilated. The anastomosis was visualized near the junction of the second and third portions of the duodenum (Fig. 3). The stoma of the anastomosis appeared small in caliber; however, the barium passed through readily and the stomach was empty in approximately one hour.

DISCUSSION

The accurate incidence of congenital stenosis of the duodenum is not really known. Ladd and Gross¹ reported 71 operated cases of intestinal or colonic stenosis, of which more than 50 per cent occurred in the duodenum. In 1922, David and Poynter² collected 392 cases from the literature. In 1931, Webb and Wangansteen³ reported an increase to 500, and since then occasional individual cases or groups of cases have been reported. The reported incidence based on autopsy material varies from 1 in 215⁴ to 1 in 20,000² autopsies. Ladd feels the clinical incidence is more common than is usually supposed.

The etiology of stenotic duodenal lesions has not been definitely established; the most feasible explanation, however, is based on observations of the embryologic development of the gut. With reference to the duodenum, the embryologic events relevant to stenosis take place in the fifth to tenth week of fetal life.⁵ Up to the fifth week, the intestinal lumen is well defined and lined with epithelium. Shortly thereafter the epithelium proliferates and occludes the lumen, the so-called "solid stage", and which persists for a short period. Later a vacuolization occurs, the vacuoles coalesce, and the lumen is reestablished. Failure of coalescence in an intestinal segment produces an atresia, whereas incomplete coalescence produces a stenosis.

The pathology of a stenosis may present two types^{1, 2} (a) a diaphragm or veil which is partially perforated, or, (b) simple narrowing of various degrees. The diaphragm may consist of the usual coats of bowel, or, just of the mucosa. The bowel proximal to the obstruction is dilated and tense; distally it is collapsed and very small.

A review of reported series of cases1, 2 which include both steno-

sis and atresias of the intestine indicates that obstruction is more frequent in the duodenum and ileum. The duodenum is the frequent site for stenosis. In about 15 per cent of cases of intrinsic obstruction the lesions are multiple, but the majority of multiple lesions² are atresias. It is rare to find more than one site of obstruction in cases of stenosis. It is important to emphasize that conditions such as an annular pancreas encircling the duodenum, herniation through a mesenteric defect, or constricting mesenteric bands cannot be assumed to be the only cause of obstruction until a patent lumen is demonstrated.

It has been noted that a few babies with congenital duodenal obstruction are Mongols, an observation which has not been made with intestinal obstructions at other levels. We have had

one such case with duodenal atresia.

The clinical picture is primarily that of persistent vomiting.^{1, 3}

About 50 per cent of the newly born present this complaint in the first or second week of life. In atresias, the symptoms make their appearance within the first few days of life, whereas stenotic babies' symptoms are dependent upon the degree of stenosis. Sometimes symptoms may be delayed into infancy or childhood, where the symptoms consist of episodes of abdominal cramping associated

with vomiting.

The vomitus usually contains bile except in those cases where the duodenal obstruction is supra-ampullary. Even in the latter, anomalous bile ducts above the obstruction may produce bile in the vomitus. Distention of the abdomen is present in lower stenosis or atresias and also in duodenal lesions, where it is chiefly in the epigastrium, providing the stomach has not been emptied by vomiting. Peristalsis may be visible. The stools are usually quite small in amount, dry, grayish-green in color, and even may be indistinguishable from meconium. Examination of the stool for cornified epithelial cells may be of value in this situation. The baby usually is dehydrated secondary to the vomiting and runs a low-grade fever.

Confirmatory evidence of the diagnosis and assistance in locating the obstruction may be obtained by x-ray examination, placing the infant in upright and inverted positions to show the extent of the gas-filled intestinal loops. Barium should be avoided if possible. Radio-opaque oil may be employed as a substitute. Proximally the duodenum is greatly dilated and distally no radio-

opaque material is visible, although some may appear in stasis films indicating an obstruction or inadequate luminal diameter at the site of the stenosis.

The treatment of congenital duodenal stenosis should be aimed at the restoration of fluid and electrolyte balance and the correction of the deformity by operative methods. Gastric aspiration every four hours is preferable to an indwelling Levine tube, which is often poorly tolerated. The restoration of the fluid balance should be undertaken very carefully with judicious use of saline, since salt loss by sweating is so much less in the newborn. Anemias are corrected by small transfusions.

The anesthesia of choice is open drop ether, although several experienced pediatric surgeons employ cyclopropane.⁵ The use of an intratracheal tube and curare is advocated by some surgeons. It is probably best to choose the anesthetic based on the personal experience of the surgeon and the availability of a well-trained anesthetist.

The operative technique includes a liberal right rectus incision, although the oblique transrectus incision will give excellent exposure to all parts of the duodenum. The dilated loop proximal to the obstruction presents itself fairly readily, beyond which the bowel is often of minute size. At the time of surgery we have found it helpful to feed a small Levine tube down to the site of obstruction. By alternatively deflating and inflating with small amounts of air, the site of obstruction can be seen or felt more readily.

If the stenotic area is above the ampulla of Vater, one must resort to a gastroenterostomy, but for the usual duodenal stenosis a side-to-side duodenojejunostomy is the procedure of choice. A gastrojejunostomy is technically easier, but the results are not as good, 9. 3 possibly because bile may be regurgitated into the stomach and neutralize the acidity.

Ladd⁹ prefers a retrocolic duodenojejunostomy, although Donovan¹⁰ reported five cases successfully treated by anticolic anastomosis.

Morton and Jones¹¹ have employed a different technique, which is essentially a plastic reconstruction after removal of the velum or diaphragm. This consists of opening the bowel in longitudinal fashion, destroying the velum, and suturing the bowel up transversely. In general, these procedures are ineffectual and disap-

pointing and perhaps should be discarded entirely. The anastomosis of the dilated proximal duodenum to the jejunum offers difficulty because of the small size of the jejunum, but a side-to-side anastomosis using a two-layer suture technique is satisfactory. It is perhaps best to keep the duodenum deflated at this stage by

an indwelling Levine tube for a few days.

The survival rates following surgery for duodenal stenosis up until recent years have been discouraging. As was stated previously, it is difficult to ascertain if reported cases were actually atresias or stenosis. The reported results show that high obstructions stand a better chance of survival than those below the duodenal area. Since stenosis apparently occurs more frequently in the duodenum than does atresia, this may account for the better prognosis in cases of stenosis. In Ladd's cases1 of duodenal stenosis for the years 1940 through 1952, twenty-one patients were alive and seven dead.

In summary, two cases of duodenal stenosis are presented. The surgery of duodenal obstruction is discussed and the importance of differentiating between duodenal stenosis and atresia is pointed

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- 8.

PHYSICAL THERAPY AND REHABILITATION FOR MENTALLY AFFLICTED CHILDREN

HYMAN GOLDSTEIN, M.D., Sc.D. New York.

No problem in the medical field to-day is more in need of thoughtful attention than that of the mentally afflicted child in whom the pyramidal and extrapyramidal pathways are affected, thereby weakening the voluntary muscle power and altering its tone and spasticity. This field is a most challenging one, so much so that we must be concerned in advising troubled parents wisely, relieve them of considerable anxiety, worry, and as much expense as possible and, at the same time, we should administer to their affected children properly directed physical therapy and rehabili-

tation by trained physiotherapists.

Encephalopathies in children which result from damage to the cerebral cortex, cerebellum, corpus striatum, brain stem, basal nuclei, neuroglia and other areas of the central nervous system, give rise to pyramidal and extrapyramidal pathway lesions to a variety of cerebral palsy syndromes and symptoms. Any disease process or injury to the brain of a child which can adversely affect either the motor system proper such as the pyramidal and extrapyramidal pathways, or the cerebellar systems, or both may be a cause of cerebral palsy. During intrauterine life, incidental x'radiations, deep anesthesia to the mother (safe to postpone unimportant operations), noxious maternal conditions such as endocrine disturbances, negative nitrogen balance from lack of daily protein intake and general malnutrition, intoxications, infections, genic germinal defects, disturbed placental circulation and placenta defects, trauma, Rh and blood group incompatibility and blood dyscrasias, may cause the infant to be born with immature cerebral gray and white matter or arrested brain developmental lesions. cerebral anoxia giving rise to immediate and residual brain lesions and symptoms, and severe brain injuries that may subject the newborn baby later in life to a miserable existence, especially, if left untreated. Paranatal factors such as prematurity, trauma, physical injury, and asphyxia neonatorum are important causes contributing to cerebral palsy. During delivery mechanical stress and release causes circulatory cerebral defects endangering life and health of the infant. Dr. Philip Schwartz1 reported the following, the principle types of late cerebral palsy sequellae. 1. central and peripheral porencephaly; 2. sclerosis of the white matter; 3. sclerotic microgyria; 4. cicatricial basal ganglia including caudate nuclei defects; 5, status marmoratus; 6, hemorrhagic internal pachymeningitis; and, 7. internal hydrocephalus. Other lesions are cortical hemiatrophy, cerebral agenesis, cerebellar lesions, brain cysts, small areas of yellow necrotic foci of the brain, immaturity and demyelinization processes, arachnoidal adhesions, and venous angiomata, aneurysm of small arteries or veins, and congenital brain anomalies. Such an array of cerebral and central nervous system pathology explains why we have a variety of cerebral palsy syndromes such as spastic, athetotic, tremor, rigidity, flaccid or atonic, and the mixed types. About 60 per cent of them are complicated by mental retardation, and from 25 per cent to 40 per cent with epileptic seizures. The prognosis in each case depends a great deal upon the child receiving the proper treatment which in many children will improve many of the crippling symptoms. The personality of the treated child is improved too.

Historically, descriptions of crippled and lame children date back many centuries. Felix Wurz,3 in 1696, wrote a children's book about malformed and lame children; Cazauvielh,3 in 1827, described brain atrophy; Cruvielhier, (1829-1841) reported cases of left cerebral hemiatrophy and microgyria; Herschl,5 in 1859, described porencephaly; Dr. Wm. J. Little,6 in 1861 reported a case of cerebral diplegia which is known as Little's Disease; Virchow, in 1867, described cystic degeneration of the centrum; Jendrassik and Marie,8 in 1885, described cerebral hemiatrophy; hydrencephaly by Schaeffer,9 in 1896; spastic feet deformities by Meissner,10 in 1850; microgyria by Kotschetkowa,11 in 1901 and degenerations of the cerebral white matter by Schilder. 12 in 1912. Deformed spastic children became the artists favorite study during the Renaissance period, and later, paintings of spastic children appeared by Raphael, Cigolius, Piola, Poussin, and others. This, of course, stimulated the interest of the medical profession and research workers.

The spastic form of cerebral palsy occurs in about 65 per cent of cases due to a cortical lesion which normally controls planned, voluntary movements. The choreiform-athetotic type occurs in 20 per cent of the children. The lesion is in the basal ganglia

which normally controls automatic and associated movements and certain aspects of posture. The tremor type has a similar origin and is found in 2 per cent of the cases. The ataxic form occurs in 8 per cent of the children. The lesion is in the cerebellum which normally controls balance and coordination. Rigidity occurs in 4 per cent and is similar to the spastic cases in origin, but the lesions are more widespread. The atonic or flaccid muscular type is also part of the spastic type, and is a lesion of the pyramidal tract only. The atonic and mixed forms of palsy cases make up about 1 per cent. There are approximately 500,000 cerebral palsy children in the United States, and thousands of new cases are added each year. This gives one an insight into the magnitude and importance of the problem which faces the public and the medical profession. Team work is necessary to accomplish the best results.

PHYSIOLOGICAL ASPECTS OF CEREBRAL PALSY MUSCLES

Muscle Stretch Reflex. Normal muscles remain at rest when not in use, and are capable of contracting to the degree of tenseness that will counteract gravity. When a normal muscle contracts, its antagonist relaxes, which lowers the muscle tone enough to carry out the motion. This is known as the "Sherrington's Law" of reciprocal innervation. The spastic muscle is hyperirritable and reacts to any stimulus, especially to a stretch stimulus. Assuming the biceps muscle to be spastic, if we extend the elbow quickly from the flexed position, the biceps is stretched. This "put on the stretch" is enough stimulus to cause the biceps to contract, which may halt or only allow a slow uninterrupted motion. The degree of spasticity is recorded and the extent of the blocked motion may be measured with a goniometer or angle rule. The stretch reflex test should be applied to every muscle in the body and checked on the patient's chart as a guide for administering proper physical therapy.

Another type of muscle disorder is the flaccid or "zero cerebral" muscle designated as "O. C." muscle (O-zero; C-cerebral). The O. C. muscle is flaccid as in poliomyelitis, it is cerebral in origin belonging to the atonic muscle in the spastic type case of cerebral palsy. The test is made by the child himself being asked to move all the muscles in the body, one at a time, and any muscle that cannot contract, the therapist places under observation, such as "flaccid biceps" in elbow flexion contracted position

and he notes whether or not the little patient can hold that position. If he cannot hold this position of elbow flexion and his forearm drops to his side on the table, the biceps is recorded as O. C. It

has not the power to contract at will.

The athetoid cerebral palsy child is divided into the tension athetoid child and the non-tension athetoid. In athetosis we get muscle contractions without voluntary direction and without voluntary control. Therefore, the motions are involuntary only. When the athetoid child attempts to stop the purposeless motions or limb movements, he becomes tense and very often the contracting muscles stiffen, thereby resembling the spastic muscle. This we call tension athetoid. If we shake the limb when in an athetoid tension contracted motion, it relaxes while the spastic muscle becomes more tense. In this way we can differentiate between the tension choreiform-athetoid movements and the spastic child. The athetoid child who makes no attempt to stop the involuntary movements is considered to be a non-tension athetoid. During excitement caused by loud noises, talk, or the presence of many persons nearby, a spastic child will become more spastic while the athetoid child will not increase tension motions. This is another means of differentiating between the two types of cerebral palsy.

Overflow muscle reaction is another type of muscle motion, and it may be normal or pathological. Spastics and athetoids exhibit pathological overflows. A stretch reflex occurring in a muscle may produce additional stretch reflexes which we call pathological overflow motions usually on the same side in distant parts of the body. Purposeless elbow movements may excite overflow motions to the knee and ankle of the same side. Normal overflow motions are found in normal children, i. e. a simple act such as opening a difficult door may stimulate muscle contractions elsewhere, as facial grimaces, and other excess motions-body twisting and flexing of the knee, or tightening of other distant muscles, none of which have anything to do with the act of opening the door. These additional excess motions are called normal overflow. The same phenomenon is in evidence when a child lifts a weight from the floor to a table. Pathological overflow is continuous with the athetoid patient. It is the outcome of a stretch reflex of a muscle produr ing additional stretch reflexes to muscles elsewhere in the body.

Rigidity is indicated in the child who gives evidence of stiffness in the leg and arm muscles but has no involuntary motions or stretch reflexes present. Automatic or confused motion is a condition in which a flaccid muscle (zero cerebral type) when stimulated by the therapist causing resistance to active muscle contraction in another muscle, that is, if we press manually against the right hip and thigh and ask the child to flex the thigh or the hip against this resistance, we notice that the O. C. or zero cerebral dorsiflexors of the foot on the same side will contract and dorsiflexion will take place. Knowing this, we utilize this confused muscle motion contraction, also known as automatic motion, as a muscle therapy technic. If dorsiflexion of the foot fails as it does in some instances, then corrective tendon surgery is indicated.

The instructor should be trained to recognize disturbed physiological muscle stretch reflexes; zero-cerebral flaccidity; normal and pathological overflow motions; automatic and confused motions; rigidity; muscle blocks or spasticity; clonus and maintained clonus; and, disturbed joint motions. This requires a great deal of patience and practice. After the therapist applies these muscle tests to a child, he should record the results on a muscle diagnostic chart which should be attached to the patient's record as a guide to planning the physical therapy exercises and muscle training program for each case. It is also essential for the trainer to acquaint himself with the personality characteristics of the patient which varies in each type of cerebral palsy. For the most part the cerebral palsy patient is handicapped by his own character, and unless his personality is molded through training, the desired goal will not be reached. Therefore, the proper approach by the therapist to each case is important. The spastic child is a shut-in type, an introvert while the athetoid is fearless and an extrovert. The former requires a great deal of careful handling to develop trust and confidence in the trainer. The latter type demands praise, love, and devotion. The ataxic child is more like the athetoid but less fearless. The wrong approach may create situations which might slow up training.

TREATMENT

The physical therapy or training period for the cerebral palsy child is a long, slow, and tedious one. The parents should be made aware of this. The aim of the treatment is to give the little patient a happier adjustment to life, to make him help himself, and become useful to society. We wish to rehabilitate him so that he will become self-supporting, more or less, and develop the ability of self-help so that he will not require constant attention from another person. The severity and degree of handicap varies in each case and should be studied before starting treatment. A spastic child may have a monoplegic, diplegic, triplegic or quadriplegic involvement. If we consider the child, not as he is now but as he will be ten years later, and what his capabilities will be at that time, the treatment accomplishments will be much greater. Before touching on physical training methods proper, I will describe briefly some of the prophylactic measures for the prevention of cerebral palsy, and in many instances mental retardation and other complications.

Dr. William Sharpe¹³ reported, in 1928, and in 1952, a means of preventing small brain hemorrhages in the newborn from becoming potential cerebral palsy cases by performing diagnostic and therapeutic spinal taps on babies during the first twenty-four hours of life. In 500 babies, 9 per cent of them showed bloody spinal fluid which cleared after a few therapeutic spinal taps. None of the children developed cerebral palsy after a follow-up study of seven years. Another precaution against cerebral hemorrhage in the newborn is the intramuscular injection of synkayvite 5 or 10 mgm, into the pregnant woman before delivery which may prevent fetal cerebral bleeding. Prevention of noxious states in the mother will help, such as endocrine balancing therapy during pregnancy. Pregnant women should avoid contact with sufferers of virus or contagious diseases. If a woman contracts a contagion, intramuscular injection of immune globulin into the expectant mother may prevent the virus from attacking the fetus through the placenta partition. In young or grown-up children who contract meningo-encephalitis from pneumonia, meningitis or from postmeasles, chicken-pox, vaccinia, virus infections, or following pertussis vaccine injection leading to brain lesions, we may avoid the cerebral complication of mental affliction and mental retardation therefrom by administering intramuscular injection of 0.14 cc. immune globulin, per pound of body weight, to the child as early as possible. Another preventative measure described in 1954 by the author14 is the typhoid vaccine hyperpyrexia regime by Bower and

Knouf of the Los Angeles County Hospital for Children, reporting a series of 120 cases of post-viral and infectious encephalitis with successful results.

TREATMENT PROPER

When the examiner records the condition of a child's muscles in accordance with the Standard Lovett System for muscular grading when the contractions are left, moving the limbs or any part and/or joints through a partial or full range of motions against gravity, or with gravity eliminated by having the child lie on his side and grades them as zero, trace, poor, fair, good, and normal muscles, he is ready to plan a training schedule for the child. In this respect I follow the ideal set-up of modalities introduced and used by Dr. Winthrop M. Phelps for cerebral palsy children at the Children's Rehabilitation Institute at Baltimore, Maryland and also followed up by Miss Paula F. Egel¹⁵ and staff at the Children's Hospital, Buffalo, New York.

A modality which is a specific phase of treatment, is used exclusively for cerebral palsy conditions. There are 15 modalities for the treatment of all types. 1. Massage; 2. Passive Motion; 3. Active Assisted Motion; 4. Active Motion; 5. Resisted Motion; 6. Conditioned Motion; 7. Automatic or Confused Motion; 8. Combined Motion; 9. Rest; 10. Relaxation; 11. Motion from the Relaxed Position; 12. Balance; 13. Reach and Grasp; 14. Reciprocation; 15. Skills.

Massage. This is an important modality of mild massage and muscle kneading which I use for strengthening the weakened muscles in ataxia and the zero-cerebral or O. C. muscles in the atonic spastic type. It is rarely used in the other types of cerebral palsy children.

Passive Motion. Important for teaching smooth motions, flexion, extension, adduction, abduction and circumduction in extremity and joint muscle re-education without disturbance of the stretch reflexes in the spastic child. The athetoid patient first must learn to relax to quiet involuntary movements, sometimes with the use of sand-bags, then by passive motion. This is true for the tremor cases also. The ataxic child lacks the kinesthetic sense which can be developed by passive motion exercises. For the rigidity type case, this passive motion is applied at a faster rate of speed.

Active Assisted Motion. We try to teach the children how to

relax and then go on to passive motion providing the involuntary, purposeless motions and overflow muscle contractions are under control. If these children can relax and remain quiet, then active assisted motions are good for them. With this modality they learn how to use their muscles properly, and it becomes the educational beginning of activity or functioning of the muscles. The child should not receive help nor resist gravity. Therefore, the child should lie on his side when he receives this exercise. Appliances in use such as crutches as an aid to prior modalities should be continued and gradually removed so that the arms and legs eventually will move without these aids. This modality is most important for the ataxic type, to prepare him for future training; the spastic, athetotic, tremor, and rigid types do well under it.

Resisted Motion. During the time we use this exercise one should be sure that the mattress sheet or cover, the linens and pillow on the table, are smooth. Wrinkled sheets interfere. Illfitting braces interfere too. On the other hand, the trainer should make use of resisted motion manually against the part of the body that is being exercised actively by the child. For example if we wish to produce resisted motion to the triceps in the arm, one exercises force against the forearm while the elbow is being straightened. It slows up the triceps muscle motion. The degree will depend upon the amount of resistance used by the trainer. It helps to strengthen the antagonistic muscles to the spastic ones giving them more power. It also strengthens muscles weakened by disuse. It is an excellent modality of therapy for the zerocerebral muscles or flaccid muscles. It is an excellent method in use for forced chair sitters. It educates and strengthens the function of weak muscles. We should work against gravity to get more resistance, with feet dangling off the table, or with the child in a supine position. Stretch reflexes and overflow movements should be eliminated first. The athetoid motions are joint movements rather than involvement within the muscles but it may be used to clarify the feeling of the muscle action during involuntary motion. It is excellent for the ataxic type where all the muscles need strengthening. The other forms may be aggravated by this modality except when used cautiously, where indicated.

Conditioned Motion. Following the principle applied to the Pavlov dog where the conditioning impulse was developed in the dog's stomach by ringing a bell when serving his meal, his gastric juice would pour out every time the bell rang even though food was not being served. So in cerebral palsy children conditioned motion can be stimulated by singing a simple rhyme with each combination of exercises. After weeks of repetition the child will repeat the same exercise every time the associated rhyme is sung by the trainer. Rhymes were first used by Miss Jenny Colby in 1883, a graduate of Perkins Institute. The trainer should learn the rhymes outlined by Miss Paula F. Egel¹⁵ in her book on cerebral rhymes and motions outlined by Miss Paula F. Egel¹⁵ in her book on cerebral palsy, namely,

SONGS	EXERCISES
Shoot the rocket.	Supine: Forward flexion of the shoulder.
	Prone: Backward flexion of the shoulder.
Pump the water.	Supine: Internal and external rota- tion of the shoulder.
Sunbeams rise and fall.	Supine and prone: Abduction of the arms.
Up and down.	Supine: Flexion and extension of the elbow, etc.

There are about 20 rhymes and corresponding passive and active exercises which are easily mastered by children, and the associated exercises become automatic performances in conditioned motions. It is a good introductory treatment modality for ataxic children. It is an important and helpful method when applying passive and active assisting motions. This can be applied to all types of cerebral palsy children. We must guard against involuntary interrupted motions, stretch reflexes and overflow contractions.

Automatic or Confused Motions. We must bear in mind that there are three kinds of spastics i. e., one with stretch reflexes (spastic muscles) only, those with O. C. muscles only, and those that have a combination of both. The O. C. involvement is a type of flaccid muscle disorder that is found only in the spastic group, and which has no voluntary ability to contract, nor stretch reflex but can be made to work only if given confused motion. This confused or automatic motion is obtained when we press gently but firmly with both hands against the hips and thighs of a child who

resists any of the muscular action beneath, the foot drop O. C. muscles will be stimulated to contract and place the foot in dorsiflexion. Doing these exercises for weeks and/or months will finally give strength, and good function to these previously flaccid, cerebral palsy O. C. muscles. We must find, in various parts of the body, which constrained muscle actions in one area will stimulate O. C. muscles in another, usually on the same side of the body. We must begin with a maximum amount of resistance to obtain this confused motion which is based on the principle of pathological overflow. I cite two cases in point. A child of 6 years of age suffered from spastic diplegia with a typical cross-legged scissored-gait walk. Gluteus medium muscles were found to be zero cerebral in both hips. Confusion found was resistance to deltoid muscles of both shoulders.

Treatment. The child was put in a supine position on an exercise (smooth) board to assure frictionless leg motion. Resistance was applied to arm abduction. At the start the confused motion failed to work, but after several attempts more and more exercise produced a wider range of movements in the legs. After several months' training this girl progressed to constrained motion and no longer required the trainer's assistance. A wide bandage was applied to hold her arms close to her sides while standing. The resisting factor produced leg abduction while walking. The bandage was gradually loosened and finally removed permanently, with very satisfactory results in improved appearance and walking ease.

The second case was a boy with spastic hemiplegia. Zero-cerebral muscles were recorded in a 5-year-old involving the superinators of forearm, wrist extensors, and finger extensors of the left side. Confusion was found in the uninjured arm when corresponding motions were resisted.

Treatment. Supine position, resistance applied until constrained motion was substituted in training and the O. C. muscles developed so that the injured hand could assist the good hand. The boy was able to sit and stand and get around after many months of therapy.

Combined Motion. The ataxic children can use combined motions early. The other cerebral palsy types can receive combined motion technic when other modalities have freed them from stretch reflexes and overflow involuntary motions as an advanced form of therapy. Ataxic cases lack kinesthetic sense and are, therefore, unable to stabilize the muscles. In combined eye-to-hand coordination, they are taught strong tactile sense development which acts

as the child's stabilizer. It teaches two active simple joint motions that are useful in fulfilling everyday needs, i. e., finger flexion combined with wrist extension; elbow flexion combined with supination of the forearm; elbow extension with pronation of forearm; wrist flexion with finger extension; finger flexion with forearm supination. Forward flexion of shoulder with elbow extension, internal rotation of shoulder with elbow flexion and other lower extremity combination of motions,

All types of cerebral palsy children require more rest than the ordinary child. The ataxic child makes many movements at random, uncontrolled muscles contract, and all types burn up lots of energy. Where possible, they should lie down and rest. Some require braces and splints. The type of rest needed for each child and the time element is up to the trainer. Those who cannot sit because of weakened back muscles may require a corset, back-

rest, cast, or especially constructed chair.

Relaxation. This is a specialized technic, and it helps to reduce tensions. Relaxation is started proximally and proceeds distally. First we have the patient contract the muscle, then let go to relax it, first the forehead by elevating the eyebrows, frowning, then refaxing; move eye-balls up, down and to the sides, then let go; then the mouth by grinning, pursing mouth, opening and closing jaws, grit teeth, push lips together, then let go; tongue is extended retracted, elevated and depressed, then relaxed; pharynx by saying ugh and ung, breathing through nose six times, and count by whispering, then relax; neck by raising head off pillow, drop, turn head to right and left, then relax; scapula, hug shoulders, relax, push shoulders down and let go; ribs, expand chest by deep breathing, abdominal muscles relax also, breathe out deeply and let go; shoulder, lift shoulders off bed, push down on bed, roll to inside and then outside and let go after each movement; flex elbow, then extend firmly and relax, roll forearm first out, then in, and relax; wrists, flex, extend, ulna and radial deviation relax with each movement; fingers, open wide, close fist, opposition of thumb to fingers and relax after each motion; hips, lift pelvis off bed, push down on bed, push out and roll in and out, relax after each motion; ankles, dorsiflex, planter flex, invert, evert and let go after each movement; toes, curl under, straighten, close together, spread apart, and relax after each exercise.

Relaxation modality is very effective in the athetotic type child.

Athetosis disappears completely during sleep. Voluntary movements are thereby made possible without interference of the involuntary motions. Relaxation can be easily learned. Its use reduces the amplitude of athetoid motions to an almost negligible degree. The patient becomes relaxed rather than tense and improves constantly. The trainer can use a mirror so that the child can see himself relaxing. This helps. In the other types, relaxation relieves tension somewhat and helps the child in other exercises he is taking. Relaxation is a very important therapeutic modality.

Motion from the Relaxed Position. It is a method of moving, passively, different parts of the body after maintaining relaxed position. It is excellent for the athetoid patient. The child should lie on its side to avoid gravity pull. This modality is used also for the tremor but is not given to the spastic, ataxic, nor the rigid-

ity types.

Balance. In order to maintain proper balance, muscle groups and their opponents must work with proper accord in relation to each other. This is essential for the purpose of maintaining specific positions as sitting, kneeling, crawling, standing and walking. The pull of gravity helps the bodily movements and muscular contractions and tone in proper amount while the antagonistic muscles relax at the proper speed to keep good balance. The trainer should not mention words like "fear" or "afraid" to a spastic child who is full of fears. He may not cooperate at all. The ataxic child must be trained patiently to gain and maintain a sense of balance. Balance a book on his head while sitting or standing. He can be taught trunk and kneeling balance. He may stand with legs greatly abducted, then gradually lessen abduction until he assumes the normal standing position. Leg braces and skis are an aid for training standing balance. Crutches also can be used.

Reciprocation. It is an important phase or modality in treatment of all types of cerebral palsy children. Most of the little patients do not walk before treatment is begun. They do walk afterwards. Reciprocal motion of the legs is a physical act which is ordinarily learned by the infant when he kicks his bed covers off. It becomes more highly developed when he crawls. The cerebral palsy child's handicap involves his arms and legs, in not learning these actions automatically, the cerebral palsy victim is retarded and must, therefore, be taught reciprocal motion if he is to walk. Alterna-

tion and reciprocation are two entirely different motions and should not be confused by the trainer. By reciprocal motion, we move one extremity up and as we move it down the other side is moved up and so we repeat the reciprocal up-movement of one lower extremity while the other is moving in the opposite direction, downward etc. Alternation is accomplished by moving one extremity upward then bringing it downward to a resting position. When it assumes its resting position, the other leg will be raised and lowered in the same way. Walking between parallel bars teaches reciprocation. The child may be wearing braces. The slip grip may be used on the bars so that the hands can slide along and keep the child from falling. He is made to feel secure and confident. The abductor board can be used to keep feet separated while walking. One can use foot twisters if necessary to correct eversion or inversion of the feet. The legs can be exercised in the supine and prone position on a table. Skis have a double advantage in correcting faulty conditions of the feet, and reciprocation may be developed. A head helmet protects the child from injury in falls, and at the same time the child loses fears and gains confidence. Leg braces with knee extension cuffs may be used to good advantage in this training. Hip extension and flexion is also reciprocal. Walking with a tripod crutch is helpful by teaching a reciprocal hip pattern to the child. In most cases the use of the ordinary walker is harmful, making the gait awkward without muscle movement regularity or rhythm.

Reach and Grasp Movements. This modality is as important to the arms as reciprocation is to the legs. It is the last practical step in the physical therapy treatment for all types of cerebral palsy children and deals with the upper extremities only. Reach and grasp is learnel by the normal infant about the same time he learns to kick his covers off. This exercise teaches the palsy child how to use his hands and arms, since it requires a combination of joint motions. But first the child must master a single joint performance. The sitting position is best for this performance. The trainer begins with passive motion of two, then three, and four movements until all the arm and hand motions become coordinated making reach and grasp possible. It establishes a joint consciousness by the patient. Tenseness may be necessary. When performing ordinary forward flexion of the shoulder, it is necessary to tense the rotators in order to keep the humerus from rotating.

This tensing of other muscles is called a combined motion. One side of the body leads and the other side assists. The main purpose of this modality is to teach the child how to use his hands and fingers for the act of grasping and releasing. The children are pre-

pared for feeding, dressing, toilet habits, and even typing.

Skills. The teaching of skills to the cerebral palsy child, especially the retarded child, follows the reach and grasp modality and is the final step in their treatment. A skill is taught to the patient after we feel certain the child performs extremity joint motions with ease and control. The simple skills should be taught first. If sitting requires help, the skill should be started in a lying position and progress to a relaxed sitting position. It is easier to get results from the athetoid and non-intention tremor case than from the spastic and the ataxic. Sandbags may be used to obtain muscle control, then should be removed as soon as possible. Teach a child to lace a shoe. Have the child first work on a large wooden shoe nailed to a board, then try him on a smaller shoe and, if successful, have him lace his own shoe. The steps of progression should be gradual so that control may be maintained. The act should be repeated once daily. The patient should learn the skills required to make him self-sufficient in feeding, dressing, cleanliness and personal neatness, brush teeth, brush and comb hair, writing and typing, toilet habits, and later, when capable, he should be taught a trade, with self-support as the goal. Children should gradually be made to realize that they are being trained in a skilled vocation, first in a sheltered workshop, and then, when ability is shown by them, to be transfered to a regular trade school so that each child can be trained for a specific qualified trade, and later secure employment and earn partial support. They should receive praise for their successes. The child should be made to feel these acts are outstanding feats of performance. The job of teaching a cerebral palsy patient to become self-sufficient is difficult, painstaking, and long, but when completed, it is rewarding in itself. Children who cannot walk are made to walk; those who do not talk sometimes are taught speech, and a great majority of them learn many skills. A great responsibility rests with the trainer. A cerebral palsy clinic as well as a qualified children's mental retardation clinic should be well equipped with proper apparatus and qualified personnel and medical staff.

Apparatus Equipment. It is essential to have the proper equip-

ment and apparatus to work with in a rehabilitation clinic. We should have padded 7" diameter exercise rings used for grip development. Constructed chairs with commode, side arms, sloping back, head rest and foot holds are useful aids for the severe type of cerebral palsy child with weak back and hip muscles. They may need a Taylor two-bar back brace, or, a Knight four-bar one in addition to a leg, ankle, or fixed night ankle brace, to learn how to stand and walk. Splints, casts and slings are essential to immobilize limbs where necessary. Straps and weighted boards should be on hand for correction of heel walking; skis are important for ski-walking to develop anterior-posterior and lateral balance; to correct toe walking, as well as eversion and inversion of the feet. Standing tables with a cutout and supporting back helps the child to obtain his standing position in training. He may have to wear his braces until he learns how to stand and walk. A treatment table should be on hand measuring 72 inches in length, 36 inches wide, and 32 inches high covered with a smooth mattress and pillow. Tricycle and bicycle on platforms could be used for balancing and muscle development exercises. For spastic and weak muscles, especially of the fingers and hands, machine muscle activators can be used to prevent contractures, and stretch or correct early contractures. Twisters one inch wide and shoe buckles are very handy to twist around feet and legs up to the thighs sometimes to the waist, to correct abnormal foot positions. Webbing strips 12 inches wide and 27 inches long are used to sew around standing bars and fit the webbing enclosure around the limbs as knee braces to help the child learn to stand while muscles are being strengthened. Other aids are sandbags, pillows, sheets, weight scale, pelvic binders, stools, kleenex, surgical instruments, thermometers, and sundry supplies; a caliper measure in centimeters, and a goniometer to measure angles; pulleys, various weights and overhead movable frame attached to the treatment table are essential for muscle and limb stretching. Ramps, roller skates, shoe modification charts, relaxing chair, and polished exercise board are aids for exercises. For walking balance, training parallel bars, and an abductor board 20 inches wide, 7 feet long, with a perpendicular board 3/6 inch wide and 15 inches high across its center permits the child to walk with one foot on each side of the center board in good walking balance. This board also can be used with the aid of small, medium, or large size parallel bars depending upon age of the child. An

Alexander rubber ball can be used for head balancing exercises and body balance. All length bone and elastic corsets are in use for sitting training. Canes and crab three point base crutches are useful supports in standing and walking lessons. Mirrors give children more confidence enabling them to see what they are doing during training exercise. Practice stairs are helpful. Hydrotherapy and blanket strips for hot, wet-packs are excellent for relaxing spasticity, and softening and improving muscle tone in stiffened or rigid muscles. The UT-4 Burdick portable ultrasonic, therapy apparatus is helpful to stretch and lengthen the shortened Achilles heel tendon. Its use is limited to tendon and muscle work. This apparatus must not be mistaken for the supersonic machine perfected at the Bioacoustics Laboratory, College of Engineering, University of Illinois, by Dr. W. J. Fry and his coworkers. Its ultrasound focal beam is focussed above the range of human audibility, at 15,000 cycles per second to over 1,000,000 cycles per second, to a known pathological brain area for a specific given time that dissolves or destroys pathological cells and tissue but does not effect normal cells or blood vessels of the brain. It is shortly to be used at the State University of Iowa Hospital on humans in the field of hyperkinetic disorders, such as Parkinsonism, choreaathetosis and ballism and so on. This may prove to be an excellent therapeutic agent for our cerebral palsy athetotic and emotional children with and without mental retardation. To date, the work has been carried out in rodents, cats and monkeys with very promising results. Only time will tell,

The children may need medical aid during the period of physical training. Anticonvulsants are necessary to control epileptic seizures; tranquilizers with interval blood checks to regulate emotional disturbances; histamine injections in the schizophrenic children for control; endocrine, vitamin and mineral supplements and dietetic regulation where necessary make the cerebral palsy children more receptive and more prepared for physical therapy. Hearing and vision disturbances must be corrected. Educational and vocational guidance and training develop better personality, independence, and social maturity for the cerebral palsy and mentally afficted child. This is a great aid to their physical therapy training and rehabilitation.

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317 E. 17th. St.

SEROLOGIC STUDIES ON POLIOMYELITIS CONTACTS. E. Berger, L. A. Halff and K. Weisser. (Schweiz med. Wchnschr., 86:38-41, Jan. 14, 1956).

Complement-fixation tests for poliomyelitis were made on members of 14 families in whom one member had contracted poliomyelitis. The reactions to these tests indicated that in 6 of the 14 families others had become infected with the poliomyelitis virus, although these infected members showed no paralysis or other symptoms characteristic of poliomyelitis. The serum complement-fixation tests in four families indicated no additional infections, whereas in four others no definite statement was possible because the period of observation was too short. Complementfixation tests were also made on 27 children, from 4 to 7 years of age, who attended the same kindergarten as one of the poliomyelitis patients. The reactions indicated that infection had definitely taken place in 7 and probably in 8 others of the 27 children. The authors deduce that the virus of poliomyelitis is readily transmitted, since in more than half of the young and presumably nonimmune contacts there developed complement-binding antibodies, the formation of which must be attributed to a new infection with the poliomyelitis virus.—J. A. M. A.

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

CONGENITAL TUBERCULOSIS

MARTHA WOLLSTEIN, M.D.
Pathologist at Babies' Hospital,

As the cases of congenital tuberculosis recorded in medical literature are not numerous, the report of the pathology of the following case seems warranted.

A woman died in the Lying-in Hospital of advanced tuberculosis six days after the birth of a male child, in the eighth month of pregnancy.

The placenta measured 16 cm. in diameter and 3 cm. in thickness; its cord was implanted eccentrically. There was no marginal rim of infarcts and no larger infarct at any point. A triangular area measuring 5 cm, by 7 cm., with its base at the placental margin and its apex near the insertion of the cord, was yellow in color, soft in consistency and suggested cheesy material. There were two small yellow tuberculous areas in another cotyledon. Greyish nodules were numerous over the membranous surface, but, on comparison with a normal placenta, these did not seem to be miliary tubercles. The triangular cheesy area extended to and involved the membranes, the maternal surface of which was more roughened here than at any other point. The fetal surface of the membranes was apparently roughened, as though covered with a fibrinous exudate, over this area of 2 cm. by 3 cm. in diameter. Over the other two cheesy masses the fetal surface was in a similarly roughened condition,

Smears from the yellow area stained with carbolfuchsin and Gabbet blue showed many tubercle becilli, some of them in small groups of four or six.

On microscopic examination of the placental sections the chorionic membrane was found to contain areas of necrosis in which nuclear fragments were numerous, and few cell bodies were preserved. The central portion of these areas resembled cheesy material, and giant cells in small numbers were found at the edge of the cheesy centre; epithelioid cells were not present. The areas were irregular in shape, passing gradually into the normally preserved chorion without any limiting zone, and involving the amnion, whose covering epithelium was completely destroyed and its free (fetal) surface covered with granular fibrin and nuclear fragments. Large vessels in the chorion contained thrombi of agglutinated red-blood cells, staining bright red with eosin and not always filling the lumen of the vessel completely.

Many intervillous spaces contained fibrinous masses similar to those seen in normal placental sections. In other cases the spaces contained masses which were more homogeneous, almost hyalin in appearance, and surrounded by leukocytes whose nuclei were almost all fragmenting. These masses either filled the space between two villi completely or touched only one villus. But where they came in contact with the villus, the covering epithelial cells were wanting, although present immediately beyond the point of contact. This point is of interest regarding the question as to whether or not a normal syncytium is an efficient bacterial filter, and thus a protection to the fetus. Certainly the agglutination thrombi had destroyed the syncytium in this case, while in no section were the epithelial cells affected over a villus whose adjacent spaces were empty, or filled with normal blood cells, or with fibrinous masses.

Many villi were normal. Some contained small cheesy tubercles in their stroma, beneath an adherent thrombus, and others were fused together into necrotic masses in which the villous outlines were more or less completely lost, cheesy matter and a few giant cells occupying the centre. These tuberculous areas were readily distinguished from small white infarcts, of which a few were present in the section. Tubercle bacilli were found in the tubercles in the chorion, in the cheesy masses in the villi, and in the intervillous spaces both free and in the thrombi. Warthin and Cowie call attention to agglutination red-cell thrombi in the intervillous spaces of the placenta in their case.

Schmorl and Geipel have recently described placental tuberculosis as occuring in four forms, dependent upon the localization of tubercle bacilli: On the periphery of the villi, when the tubercles form in the intervillous spaces; in the interior (stroma) of the villi; in the the basal decidua; in the chorion, involving the amnion as well. The first form is the most frequent one, and occurs especially in placentas which are fully formed; the third variety, on the other hand, occurs in the early months of pregnancy. The second variety is very rare as a primary placental lesion (Schmorl having found it but once), though, as in our case, tubercles in the substance of the villi often occur secondarily to their formation in the intervillous spaces. The rarity of this form of primary placental tuberculosis would argue against the villus involvement while the syncytium remanis normal. So that the case here described illustrates the intervillous chorionic localization of tubercle bacilli, with secondary involvement of the villus stroma. Schmorl describes one case in which the tuberculous area in the chorion had perforated the amnion and tubercle bacilli were found on the surface of that membrane. Thus, says Schmorl, there is the possibility of gastrointestinal infection of the fetus by means of tubercle bacilli in the liquor amnii. In this connection, the case of Herrgott and Haushalter in which guinea-pig inoculations with amniotic fluid gave positive results is of great interest; and it is to be regretted that no such fluid was obtainable in our case, where the placental lesion points to infection of the amnion and liquor a nnii.

The umbilical cord showed no tuberculosis on gross or microscopic examination. Nor were tubercle bacilli found in the vessels of the cord in the sections.

As no autopsy was permitted, the mother's uterus was removed through the vagina three hours after death. The uterus measured 16 cm, in length and was very flabby. Its peritoneal surface was covered with young, translucent tubercles in large numbers. On opening the organ a large shred of apparently necrotic placental tissue presented itself. This was but slightly adherent, and beneath, as well as around it, there were cheesy areas varying from 1 cm. to 5 cm. in diameter. There were several small cysts on the cervical mucosa, but no signs of tuberculosis were visible. The placental site in the upper portion of the anterior uterine wall was covered with small clots and shreds, of which that above mentioned was the largest. The tubes and ovaries were normal in size and appearance. Tubercle bacilli in large numbers were readily found in smears from the endometrium and from the large necrotic mass.

Microscopic examination showed the uterine lining to have the structure of the decidua at term. At many points cheesy degeneration had occurred throughout its entire thickness, extending into the muscle coat to an irregularly varying depth, while small tubercles with cheesy centres were scattered throughout the middle and inner muscular coats, even at some distance from the cheesy endometrium. Tubercle bacilli were demonstrated in the cheesy areas and also in decidual sinuses which were not adjacent to a chersy mass.

Autopsy on Infant. The male child lived nineteen days. At autopsy its body was small and poorly nourished. It presented no skin lesions. The dried cord stump was still adherent.

Brain. No hemorrhage and no tubercles. Ventricles normal. Heart. Valves normal. Foramen oval, open. Muscle anemic. Lungs. No pleurisy, no atelectasis. A few small bronchopneumonic areas were scattered through both upper and lower lobes. In the right upper lobe, near the upper part of the posterior border, were two small, gray tubercles, each 3 mm. in diameter. No other tubercles were present. The bronchial lymph nodes were not enlarged; red in color.

Spleen. Weighed $8\frac{1}{2}$ grams and measured $4\frac{1}{2} \times 2$ cm. It was firm, dark red, and showed small, grayish points on the surface and in its substance resembling miliary tubercles.

Liver. Was moderately fatty and deeply congested, but not enlarged in size. Small tubercles were scattered over the surface and throughout all the lobes. One cheesy tubercle of 2 mm. was present in the right lobe. The round ligament contained fluid blood. No enlarged lymph nodes in the hilus.

Stomach. Normal.

Intestines. The mucosa in the colon and lower ileum was congested. The Peyer's patches and solitary follicles were swollen, but none were ulcerated. Mesenteric lymph nodes were enlarged, but not cheesy.

Peritoneum. Contained no fluid. The mesentery was studded with small grayish nodules which outlined the lymph vessels and resembled tubercles.

Kidneys. The boundary zone of each kidney contained from three to five small tubercles. No other lesions present. Weight, 17 grams. Suprarenals and pancreas normal.

Anatomical Diagnosis. Acute miliary tuberculosis of lungs.

liver, spleen, (?) kidneys and mesentery (?). Bronchopneumonia. Fatty liver. Hyperplasia of lymph nodes.

Microscopic Examination. Lung. Tubercles with cheesy centres and surrounded by monocular round cells, but no giant cells were found around small blood vessels. The surrounding lung tissue showed alveoli filled with desquamated epithelium, fibrin, and pus cells, which also infiltrated the alveolar walls. All blood vessels were deeply congested.

Spleen. No tubercles could be demonstrated in any section.

Liver. The tubercles were very small and devoid of giant cells. They were situated at the periphery of the lobules around the portal vessels. The connective tissue was nowhere increased and the bile ducts were normal.

Kidneys. The tubular epithelium showed a moderate amount of parenchymatous degeneration, and in the boundary zone around a blood vessel two small tubercles were present.

Mesenteric Lymph Nodes. Showed hyperplasia of their lymphoid cells and of the lining cells of the sinuses, but no tubercles were present. The blood vessels were congested. No bacilli found in any sections.

Colon. The covering epithelium had disappeared. The glands were normal and the solitary follicles congested, but showed no signs of tuberculosis.

It is evident that the tuberculous infection in this case was hematogenous in character. From the gross appearance of the mesentery at autopsy, it seemed probable that, in addition, there had been infection by means of the amniotic fluid. Such was apparently not the case, though again it is to be regretted that no animal inoculations were made with the mesenterics.

Smears from the heart's blood and umbilical vein were negative for tubercle bacilli. In smears from the liver a few tubercle bacilli were found after looking through a number of slides.

A portion of the right lobe of the liver was removed by sterile instruments into a sterile, glass-covered dish and cut into small pieces with sterile knives. The fluid thus expressed was injected into a healthy guinea-pig. For forty-eight hours afterward the animal seemed ill, but then recovered its appetite and seemed lively. A nodule appeared in the right groin near the point of injection, and the pig lost weight. On the thirty-fifth day it was chloroformed to death. The node in the right groin measured

2 x 5 cm. in diameter, and had a softened cheesy centre, smears from which showed large numbers of tubercle bacilli. Smaller tubercles were found on the parietal peritoneum near the largest node. The spleen was much enlarged and studded with cheesy tubercles. The liver contained many gray tubercles. None were present in any other viscus. Sections from the liver and spleen confirmed the evidence of tubercles in these organs; tubercle bacilli were also found in them.

Literature. The first undoubted case of congenital tuberculosis was described by Johne in 1885, and occurred in a calf fetus of eight months, found in the uterine cavity of a cow which died of phthisis, the uterus and placenta being normal. Since then numerous cases have been described by other veterinarians, until in 1898 more than 60 cases of calves with congenital tuberculosis had been reported, and Klepp estimated that 2.63 per cent of all calves born of tuberculous cows are tuberculous themselves.

Human cases of congenital tuberculosis are much less frequent, Schmorl and Birsch-Hirschfeld being the first to describe the case of a seven months' fetus born of a tuberculous mother and showing tubercle bacilli in its liver and in the placenta, no histological changes of tuberculosis being present. Sabouraud was the first to describe a well authenticated case of congenital tuberculosis in which miliary tubercles were found in the liver and spleen of the infant. Doubtful cases have been reported since the year 1825, but in the absence of convincing microscopical examinations these must remain unproved. Hauser, reviewing the literature in 1898, found 18 cases which he considered as undoubted tubercular infection of the fetus, or at least as cases of "transmission of tubercle bacilli into the fetal circulation," and which he classified as follows: 9 with extensive tuberculosis of the fetal organs; 5 in which tubercle bacilli without the presence of tubercles were found in the fetal organs, and 4 cases of placental tuberculosis.

The literature has been critically reviewed within the last year by Warthin and Cowie, whose stricter classification has served to separate the undoubted from the doubtful cases, the criteria required being "the presence of characteristic anatomical changes and of tubercle bacilli, the development of the lesions within such a short time after the birth as to preclude the possibility of extrauterine infection, and the exclusion of syphilis." From this point of view the undoubted cases of congenital tuberculosis were reduced to 5, accepting 3 of Hauser's 9 (Honl, Sabouraud and Lehmann) and adding those of Ustinow and Auché and Chambrelente. Veszprémi's case has been reported since the appearance of Warthin's paper, and thus the case here reported is the seventh of undoubted congenital tuberculosis to be recorded

The 4 cases of placental tuberculosis mentioned by Hauser are all accepted in Warthin's stricter review, and 2 reported by Warthin and that of Auché and Chambrelente added. To these the cases of Runge and Warthin and Cowie, as well as our own, must now be added. Veszprémi, unfortunately, was not able to examine the placenta in his case. Schmorl and Geipel found 9 tuberculous placentas among 20 examined in cases of tuberculosis. Hauser omitted a second case of Lehmann's. There are, therefore, 20 cases of placental tuberculosis recorded up to the present time. One of Warthin's cases is of special interest, as it was one of ectopic gestation in which tubercles were demonstrated in the tubal sac and in the placenta, few tubercle bacilli being found. The fetus was between three and four months old, and no tubercles could be positively demonstrated in its viscera. The umbilical cord in this case also showed several nodules, one with a cheesy centre, the microscope picture of which resembled that of tubercles,

Finally, the 5 cases mentioned by Hauser in which tubercle bacilli without the histological lesions of tuberculosis have been found are augmented to 12 in Warthin's review, and, with that of

Warthin and Cowie, now number 13,

The lesions in the fetus of our case were very few and recent, considering that the child lived nineteen days. The fact recalls the case of Doleres and Bourges, in which the child of a tuberculous mother died five weeks after birth and no tubercles were found in its organs, although the heart's blood inoculated into a guinea-pig gave a positive result. Warthin calls attention to this case and his own in relation to latent congenital tuberculosis and immunity of the fetal tissues, the virulence of the bacilli present having been proven by inoculation into guinea-pigs. Schmorl and Geipel believe that a few tubercle bacilli may be destroyed in the infant's body, but many cause tuberculosis, usually in early infancy. It is possible, but not probable, that they may remain latent until puberty.

Summing up the pathology of our case in the light of

Schmorl's work on tuberculosis of the placenta, and considering the advanced stage of the tuberculous lesion in the endometrium, we may assume that the basal decidua was first affected, the tubercle bacilli traveling from the decidual sinuses to the covering of the villi, localizing there and causing thrombi in the adjacent intervillous spaces with destruction of the syncytium, entering the villous stroma and finally reaching the chorion. Thus all four varieties of placental tuberculosis are illustrated in this case. That the fetus was infected comparatively late (just before birth) might be argued from the early stage of the tubercles in the child, which had lived for nineteen days.

NONERYTHROBLASTOTIC KERNICTERUS OF THE NEWBORN: ACUTE FORMS AND SEQUELAE. R. Sacrez, J.-G. Juif, L. Fruhling and others. (Semaine hop., Paris, 32-596-604, Feb. 20, 1956).

Jaundice of the newborn without blood incompatibility is not rare, but it is chiefly limited to premature and debilitated babies. The authors saw 14 such cases in four years. The first symptoms appeared between the fifth and seventh days of life. Those of cerebral involvement were severe, and there were also more specific signs such as hypertonia and athetoid movements. Death is the most frequent outcome of this acute stage of the disease. Some authors have reported the mortality to be 100%; eight patients of the present series died, usually within 48 hours after the onset of symptoms. The surviving babies have severe sequelae: five of this series had an extrapyramidal syndrome and the sixth considerable mental deficiency. The pathogenesis of nonerythroblastotic kernicterus is postulated to be the following: The premature infant develops an infection and thereby increases his hyperbilirubinemia. The infection is really the expression of metabolic disorders that severely affect the nerve cells, which are already influenced by anoxia. Then the grey matter becomes impregnated with bile. It might be worthwhile to practice routine checking of the amount of bilirubin in all premature babies and to perform exchange transfusion in those whose bilirubin values are excessively elevated .- J. A. M. A.

DEPARTMENT OF ABSTRACTS

Conducted by MICHAEL A. BRESCIA, M.D., NEW YORK

PEACHER, W. G. and STORES, R. P.: CERVICAL DISK CALCIFI-CATION IN CHILDHOOD. (Radiology, 67:396, Sept. 1956).

The case of a child with calcification of the fourth cervical disk, followed for 3 years, has been presented. Although the small number of cases reported makes statistics unreliable, this condition often appears to follow an acute inflammatory process. Symptoms referable to the cervical spine-pain, limitation of motion, muscle spasm, etc.-with or without evidence of a focus of infection and systemic reaction, usually occur. Conservative and symptomatic care are generally sufficient to bring about early subjective relief with no permanent residuals. The calcific deposits have been found AUTHORS' SUMMARY. to last for varying periods of time.

BORGEN, P.H. F.; ERICHSEN, S. and STANDAL, B.: CAT AS A Possible Source of Infection in a Case of Congenital Toxo-PLASMOSIS, (Acta Paediatrica, 45:569, Sept. 1956).

Possible toxoplasma transmission from a cat in a case of toxoplasmosis congenita is reported. It is the first confirmed case of feline toxoplasmosis in Norway. It seems reasonable that pregnant women should be warned against too close contact with sick animals (dogs, cats) even in this country. AUTHORS'SUMMARY.

MÖLLER, K. L.: EXANTHEMA SUBITUM and FEBRILE CON-VULSIONS, (Acta Paediatrica, 45:534, Sept. 1956).

Of 448 cases of febrile convulsions admitted to the hospital during 1945, at least 7.6 percent (34 cases) occurred in connection with exanthem subitum. Among the children with febrile convulsions who were 12 months old or younger, exanthem subitum was the cause of fever in 16.2 percent. The 34 cases of exanthem subitum had a median age of 12.5 months, the remaining 414 children with convulsions had a median of 20.8 months. Exanthem subitum is supposed to cause convulsions, not only because of fever, but because of a specific cerebral effect. Persisting cerebral lesions may occur, resulting in new attacks of convulsions later on. THANAWALA, J. K.: SMALLPOX IN CHILDREN. (Indian Journar of Pediatrics, 23:153, May 1956).

 Of 365 cases of smallpox in children, 100 died giving a case fatality rate of 27.2 percent. Of the 100 deaths, 36 died within 48 hours.

In Bombay, a smallpox epidemic occurs every third or fourth year. The incidence of the disease is less in children than in adults, but, the mortality rate is higher among the former than the latter.

3. The incidence of the disease is greater; the disease occurs in more severe form and the mortality rate is higher among the unvaccinated than in the vaccinated.

Seasonal rise is observed in the months of January, February, March and April.

5. Penicillin prevents disfigurement to a considerable extent.

AUTHOR'S SUMMARY.

DAVIS, L. A.: THE USE OF SOLUBLE NONABSORBABLE OPAQUE MEDIA IN THE EXAMINATION OF THE GASTROINTESTINAL TRACT IN CHILDREN. (Southern Medical Journal, 49:1011, Sept. 1956).

The water soluble, nonabsorbable radiopaque media used in urological roentgenography are valuable in examining the gastro-intestinal tract of iniants. The fact that they are of low viscosity, do not inspissate, and are nontoxic makes them superior to the ordinary barium mixtures in many cases.

AUTHOR'S SUMMARY.

Nolke, A. C.: Severe Toxic Effects from Aminophylline and Theophylline Suppositories in Children. (Journal American Medical Association, 161:693, June 23, 1956).

Thirteen patients had severe symptoms of theophylline intoxication after the administration of rectal suppositories. The clinical picture was characteristic; frequent vomiting, agitated, maniacal behavior, and unusual thirst are distinguishing features. The appearance of brown, syrup-like vomitus, delirium, convulsions, and shock are minifestations of intoxication in its severest form. Four patients died. The rectal suppository is a poor means of theophylline or aminophylline administration. The unpredictability of absorption makes a fixed therapeutic regimen dangerous.

AUTHOR'S SUMMARY

UHLMANN, E. M.: CANCER OF THE THYROID AND IRRADIATION.
(Journal American Medical Association, 161:504, June 9, 1956).

A careful review of the pertinent literature and my own observations of 25 children exposed to irradiation and of the children and adolescents with carcinoma of the thyroid do not justify the acceptance of a correlation between radiation therapy and the increased frequency of carcinoma of the thyroid in youth. The assumption that the rise in carcinoma of the thyroid corresponds to the number of children exposed to therapeutic radiation is not corroborated by facts. It could be shown that in some instances children had unrecognized carcinoma at the time they received treatments with x-rays. The amount of radiation applied to the thyroid area in common therapeutic procedures is smaller than that absorbed during chest fluoroscopy. Statistically, the correlation of irradiaton and development of carcinoma of the thyroid is untenable.

Author's Summary.

PIERCE, C. M.; LIPCON. H. H.; McLARY, J. H. and NOBLE, H. F.: ENURESIS. PSYCHIATRIC INTERVIEW STUDIES. (United States Armed Forces Medical Journal, 7: 1265, Sept. 1956).

Sixty enuretic recruits and sixty nonenuretics were studied. Compared with the controls, the enuretics were physically smaller on entrance into the naval services. They were characteristically indifferent about their habits, and less optimistic about establishing vocational independence. The enuretics exhibited a wide variety of neurotic traits and special sexual fears. They were less successful in all areas of adjustment. It was significant that they had more sibling rivalries and that their parents were strict and prone to have chronic illnesss. Psychologic, social, and organic factors are presented as possible etiologic agents. The term "Peter Pan" complex is submitted to describe the personalities of the enuretic naval recruit.

BOOK REVIEWS

Conducted by
MICHAEL A. BRESCIA, M.D., NEW YORK

THE ROCHESTER REGIONAL HOSPITAL COUNCIL, By Leonard S. Rosenfeld and Henry B. Makover, Cloth. Pp. 204. Price \$3.50. Cambridge, Mass.: Commonwealth Fund of the Harvard University Press, 1956.

This book is a report written by the Institute of Administrative Medicine of Columbia University School of Public Health. It evaluates the Rochester Regional Hospital Council as a coordinating body with a uniform goal of prevention, therapy and rehabilitation to a wider scale. This would include a rural, suburban and metropolitan area. Regional organization is a future design for higher standards of health in our present culture. As a process rather than a specific form of service which embraces the community value on health, it may be adapted to meet a wider variety of needs. It may increase it's field of specialization and the facilities will be available to a greater number of people. The program of the council experimented with many health services and learned they can provide valuable resource material from which other regional programs can plan for the future. Future regional plans may measure the potentialities of the services that were rendered and the problems and obstacles that developed. Of special interest, is the education that was available to the Board of Directors. Combined meetings of trustees, physicians and other hospital administrators were conducted to help them understand the functions of the hospital and the scope of their responsibility. These busy people are selected from many walks of life to form the hospital Board of Trustees and Directors. They have the responsibility of , making important policy decisions and appointing key personnel. I recommend this book to all civic minded peoples who consider a program of education as a good investment, measured by the health of the community. Community education deserves more planning and the reader will find much thought-provoking material on developing hospital services on a regional basis.

E. MECHTA, R.N.

OUR BLIND CHILDREN. GROWING AND LEARNING WITH THEM. By Berthhold Lowenfeld, Ph.D. Cloth. Pp. 205. Illustrated. Price \$5.50. Springfield, Ill.: Charles C. Thomas, 1956.

This book has been written with a great deal of understanding and tenderness. The tone of the book is set by the author's opening words: "Children are born to their parents to be loved." The rearing of any child raises many problems which problems are either enhanced or added to by a handicapped child. The care of the handicapped child naturally presents special problems to the parents who are apt to be overwhelmed in the beginning. In this regard the author's advice is well taken: "If what they hear and read confuses them, parents should trust their own common sense and rely upon the advice of one or two experts, their doctor and perhaps another specialist in child care in whom they have confidence. They can rest assured that the natural care which they as loving parents provide for their child is much more important than perfection in doing something for the child 'just so." This book is of great value to any parent with a blind child and also to any physician who might have to counsel and guide such a parent. There are many special problems which arise in the care of a blind child that one does not ordinarily think about. MICHAEL A. BRESCIA, M.D.

INVESTIGATIONS ON THE PREDISPOSITION TO AND PROGNOSIS OF POSTVACCINAL ENCEPHALITIS. A. Herrlich, W. Ehrengut and J. Weber. (München, med. Wchnschr., 98:156-159, Feb. 3, 1956).

A total of 78 cases of postvaccinal encephalitis occurred in Bavaria in the years between 1940 and 1955. In the course of catamnestic studies, the families of 55 of these patients were visited. A search was made for endogenic and exogenic factors that might produce a predisposition for postvaccinal encephalitis. There was evidence that age was a factor. Female infants seemed particularly vulnerable, but this factor requires further clarification. The prognosis of the disease is doubtful. More than half of the 78 patients died. A large number of the survivors developed sequelae (57%). Individual patients may have a predisposition for a postvaccinal encephalitis, but predisposition does not seem the decisive factor in the onset of the disease in all cases,—J. A. M. A.

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